International Journal of Cardiology 275 (2019) 89–94

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Relative contribution of modifiable risk factors for incident atrial fibrillation in Hispanics, African Americans and non-Hispanic Whites

Eric Shulman [a], Jay J. Chudow [a], Utibe R. Essien [b], Anusha Shanbhag [c], Faraj Kargoli [a], Jorge Romero [a], Luigi Di Biase [a], John Fisher [a], Andrew Krumerman [a], Kevin J. Ferrick [a,*]

[a] Division of Cardiology, Department of Medicine, Montefiore Medical Center, Bronx, NY, United States of America
[b] Division of General Internal Medicine, Massachusetts General Hospital, Boston, MA, United States of America
[c] Division of Internal Medicine, University of Arkansas for Medical Sciences, Little Rock, AR, United States of America

ARTICLE INFO

Article history:
Received 22 August 2018
Received in revised form 26 September 2018
Accepted 8 October 2018
Available online 10 October 2018

Keywords:
Atrial fibrillation
Epidemiology
Race/ethnicity
Racial disparities
Hypertension
Heart failure

ABSTRACT

Background: Contribution of modifiable risk factors for the risk of new onset atrial fibrillation (AF) in minority populations is poorly understood. Our objective was to compare the population attributable risk (PAR) of various risk factors for incident AF between Hispanic, African American and non-Hispanic Whites.
Methods: An ECG/EMR database was interrogated for individuals free of AF for development of subsequent AF from 2000 to 2013. Cox regression analysis controlled for age > 65, male gender, body mass index > 40 kg/m^2, systolic blood pressure > 140 mm Hg, diabetes mellitus, heart failure, socioeconomic status less than the first percentile in New York State, and race/ethnicity. PAR was calculated as (prevalence of X) * (HR − 1)/HR, where HR is the hazard ratio, and X is the risk factor.
Results: 47,722 persons free of AF (43% Hispanic, 37% Black and 20% White) were followed for subsequent incident AF. Hypertension in African Americans and Hispanics had a 7.93% and 7.66% greater PAR compared with non-Hispanics Whites. Similar findings existed for the presence of heart failure, with a higher PAR in non-Whites compared to Whites.
Conclusion: In conclusion, modifiable risk factors play an important role in the risk of incident AF. Higher PAR estimates in African Americans and Hispanics were observed for elevated systolic blood pressure and heart failure. Identification of these modifiable risk factors for atrial fibrillation in non-White minorities may assist in targeting better prevention therapies and planning from a public health perspective. No funding sources were used for this study.

1. Introduction

Atrial fibrillation (AF) is the most common cardiac arrhythmia globally and contributes significantly to morbidity and mortality with an increased risk of death, cardiovascular and renal disease in patients with AF [1] as well as increased individual and national healthcare costs [2]. Both incidence and prevalence are projected to increase in coming decades [3] potentially linked to an aging population [4]. Primary prevention strategies are limited by an unclear pathophysiology of AF [5], however, several independent risk factors are modifiable, including obesity, hypertension and diabetes mellitus, which may serve as prevention or intervention targets [6].

Most AF studies have been conducted in largely homogenous Caucasian populations. Data on racially and ethnically diverse populations is limited [6]. African Americans and Hispanics tend to have higher rates of obesity and heart failure and lower socioeconomic status (SES) as well as a greater number of additional modifiable risk factors for developing AF [7–9]. However, they have a paradoxically lower incidence of AF compared to Whites [10], referred to as the "racial paradox."

As our knowledge of racial/ethnic disparities in cardiovascular diseases grows, there is growing interest in the causes of such disparities and whether they can be diminished by appropriate reduction of contributing factors. Given limited epidemiologic literature, our objective was to identify modifiable risk factors associated with incident atrial fibrillation in a large racially and ethnically diverse population. Our goal was to identify risk factors using a population attributable risk model, to better target therapies for prevention of AF from a public health perspective.

2. Methods

2.1. Study design

This study is a retrospective epidemiological study of the development of AF in both inpatient and outpatients (n = 239,741) with n = 1,239,593 cumulative electrocardiograms

* Corresponding author at: Division of Cardiology, Montefiore Medical Center, Albert Einstein College of Medicine, 111 East 210th Street, Silver Zone, Bronx, NY 10467, United States of America.
E-mail address: kferrick@montefiore.org (K.J. Ferrick).

https://doi.org/10.1016/j.ijcard.2018.10.028
0167-5273/

(ECGs) obtained at Montefiore Medical Center (MMC), a large, urban, academic medical center, between January 1, 2000 and September 8, 2013. Patients were included if their age was >45 and <95. Patients were excluded if they had AF on their initial ECG, one ECG, or incomplete covariate data. Patients were followed for a maximum of 10-year incidence risk.

Race/ethnicity was self-reported and all race/ethnic categories were mutually exclusive. We used the term race/ethnicity as Hispanics are generally considered to be a multi-racial group, composed largely of White, but also African American and other races [11]. Heart failure was identified by ICD-9 code.

2.2. Cohort population

Our cohort population consisted largely of an inner-city population in the Bronx County, NY. The Bronx County consists of nearly 1.4 million individuals with a large non-White population – consisting of a majority of Hispanics, followed by African Americans. The Bronx County is considered an underserved area with SES variables such as high school graduation rates, higher education, and per capita income well below the national average [11].

2.3. Outcome ascertainment

Diagnosis of AF was determined by ECG. MMC uses a computerized ECG system (GE Healthcare, Wauwatosa, Wisconsin) to collect, store and analyze ECGs. This system is widely used and has been validated by the US Food and Drug Administration (FDA) and meets all applicable standards for resting computerized ECG analysis [12]. The computerized system includes the 12SL® program for automated ECG interpretation, which was used in this study. To determine the presence of atrial fibrillation, the 12SL® algorithm looks for an irregular rhythm or fibrillatory waves without the presence of particular concurrent abnormal rhythms. The 12SL algorithm to detect AF has been validated in multiple studies [13,14] with a reported sensitivity of 90.8% and a specificity of 98.9% [15]. All ECGs were reviewed and diagnosis of AF confirmed by board certified cardiologists.

All other clinical variables were extracted by searching the electronic medical records (EMR) system at MMC. All variables were obtained via EMR query during the study dates.

2.4. Follow-up analysis

Follow-up started from the initial ECG. For those who developed AF, days were counted from initial normal ECG without AF to the first ECG that demonstrated AF. For those who did not develop AF, survival days were counted from initial normal ECG without AF until the last ECG without AF.

2.5. Socioeconomic status variable

A SES variable was calculated for each individual in the cohort from the EMR query. Six SES variables for each neighborhood by zip code including 1) log of median household income; 2) log of median value of housing units; 3) the percentage of households receiving interest, dividend, or net rental income; 4) education; 5) the percentage of adults who completed college, and 6) the percentage of employed individuals in executive, managerial or professional positions, were normalized (Z scored) to the New York State average. A combined Z-score of the six variables was calculated for each patient and reported by racial/ethnic cohort. A Z-score of 0 is the 50th percentile of the New York State average. Z-scores were reported as opposed to percentages to maintain a continuous distribution relative to the New York State average and for easier visual discernment as most individuals were below the first percentile. This methodology is consistent with previous literature that has used Z-scoring techniques to facilitate comparisons between variables [16].

2.6. Statistical analysis

Descriptive statistics were produced for each cohort. ANOVA was performed to examine associations between continuous variables and χ^2 tests were used to compare dichotomous variables between patients. Statistical significance was defined by $p < 0.05$.

Cox regression analysis controlled for baseline covariates: age > 65, male gender, body mass index > 40 kg/m^2, systolic blood pressure > 140 mm Hg, diabetes mellitus, heart failure socioeconomic status less than the first percentile in New York State, and race/ethnicity. Testing for interaction and a sensitivity analysis was performed.

Population attributable risk (PAR) was calculated as (prevalence of X) * (HR − 1)/HR, where HR is the hazard ratio, and X is the risk factor. PAR analysis was chosen as it takes into account both incidence and association of risk factors with disease outcome. It is the incidence of a disease that would be eliminated if exposure were eliminated. It is beneficial from a public health perspective as it aids in prioritizing health budgets and distribution of resources depending on the proportion of outcome attributed to a particular exposure.

SPSS Version 24.0 and R Studio Version 0.98.507 was used for all statistical analysis. Proportional hazard assumptions were met as verified by plotting the Schoenfeld residuals.

2.7. Role of the funding source

The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee. The research was limited to materials (i.e., data or records) that have been collected solely for nonresearch purposes (such as medical treatment or diagnosis). The submission was approved with a waiver of informed consent.

3. Results

The final cohort included 47,722 patients who met inclusion criteria, totaling 157,482 person-years of follow-up (3.3 mean years from first ECG). Within this cohort, 9352 were non-Hispanic White (19.5%), 17,807 were African American (37.3%), and 20,563 were Hispanic (43.1%). Over the study period, 4724 patients developed AF.

General baseline characteristics by race/ethnicity are shown in Table 1. Non-Hispanic Whites were older, more likely male and had a higher incidence of AF, but had lower BMI's, lower rates of diabetes,

Table 1
Baseline characteristics by race/ethnicity.

	Non-Hispanic White	African American	Hispanic	Total	p-Value
	n = 9352	n = 17,807	n = 20,563	n = 47,722	
	Mean, % (SD)	Mean, % (SD)	Mean, % (SD)	Mean, % (SD)	
Demographics					
Age (years)	69.2 (15.7)	58.7 (16.1)	57.3 (15.9)	60.2 (16.5)	<0.01
Male	48.4%	35.3%	37.3%	38.7%	<0.01
Height (cm)	166.1 (16.4)	167.7 (14.3)	162.5 (13.5)	165.1 (14.6)	<0.01
Weight (kg)	76.8 (23.6)	84.3 (25.1)	78.3 (22.4)	80.2 (23.9)	<0.01
BMI (kg/m^2)	27.8 (7.8)	30.1 (8.8)	29.3 (8.1)	29.5 (8.3)	<0.01
Systolic BP (mm Hg)	132.3 (23.9)	136.2 (24.5)	131.2 (23.5)	133.3 (24.3)	<0.01
Diastolic BP (mm Hg)	71.0 (13.8)	77.2 (15.1)	73.8 (13.8)	74.5 (14.5)	<0.01
Mean survival years	2.8 (3.0)	3.3 (3.2)	3.4 (3.2)	3.3 (3.2)	<0.01
Socioeconomic status	−1.5 (2.9)	−3.3 (2.8)	−4.3 (2.7)	−3.4 (2.9)	<0.01
Comorbidities					
Development of atrial fibrillation	16.9%	8.7%	7.8%	9.9%	<0.01
Diabetes mellitus	14.7%	25.3%	27.2%	24.0%	<0.01
Heart failure	7.4%	9.1%	7.5%	8.0%	<0.01
Treatment for hypertension	44.4%	60.3%	53.3%	54.2%	<0.01
ECG characteristics					
LVH	30.1%	42.0%	30.0%	34.50%	<0.01
PR interval (ms)	164.7 (37.3)	163.0 (31.5)	155.3 (27.3)	160.0 (31.3)	<0.01
Medication					
Beta blockers	67.7%	61.8%	57.1%	60.9%	<0.01
Calcium channel blockers	44.3%	52.9%	41.0%	46.1%	<0.01
Digoxin	15.9%	8.8%	7.7%	9.7%	<0.01

Values in table denoted as mean or number (%). BMI, body mass index; LVH, left ventricular hypertrophy; SD, standard deviation.

Table 2
Hazard ratios of different risk factors to develop atrial fibrillation.

Risk factors	Non-Hispanic White		African American		Hispanic		Total	
	HR, 95% CI	p-Value	HR, 95% CI	p-Value	HR, 95% CI	p-Value	HR, 95% CI	p-Value
CHF	1.48 (1.28, 1.71)	<0.001	2.12 (1.88, 2.39)	<0.001	2.15 (1.90, 2.43)	<0.001	1.94 (1.80, 2.08)	<0.001
BMI > 40	1.11 (0.89, 1.40)	0.357	1.27 (1.09, 1.49)	0.002	1.10 (0.92, 1.33)	0.299	1.19 (1.07, 1.32)	0.001
SES	1.13 (1.01, 1.25)	0.026	1.02 (0.93, 1.13)	0.631	1.16 (1.04, 1.31)	0.009	1.08 (1.02, 1.15)	0.009
DM	0.95 (0.83, 1.10)	0.493	1.01 (0.91, 1.13)	0.830	1.04 (0.94, 1.16)	0.440	1.01 (0.94, 1.08)	0.832
SBP > 140	0.92 (0.83, 1.02)	0.099	1.14 (1.03, 1.27)	0.009	1.18 (1.06, 1.31)	0.002	1.08 (1.02, 1.15)	0.008
Race/ethnicity	n/a		n/a		n/a		1.78 (1.67, 1.91)	<0.001

Hazard ratios for various risk factors in multivariate Cox regression model controlling for age > 65, male gender, CHF (presence of heart failure), BMI (body mass index) > 40, SES (socioeconomic status), DM (diabetes mellitus), SBP (systolic blood pressure) > 140 mm Hg and race/ethnicity (reference group as Non-Hispanic White).

Table 3
Age- and sex-adjusted PARs and 95% CIs of established AF risk factors in each stratum of race–ethnicity.

Race/ethnicity	PAR (%) and 95% CIs				
	Heart failure	BMI	SES	DM	Systolic BP
Non-Hispanic White	2.40 (1.63, 3.08)	0.62 (−0.79, 1.75)	3.31 (0.41, 5.92)	−0.71 (−2.95, 1.24)	−3.04 (−7.01, 0.54)
African American	4.78 (4.25, 5.26)	2.54 (0.98, 3.88)	1.30 (−4.19, 6.26)	0.30 (−2.59, 2.89)	4.89 (1.29, 8.15)
Hispanic	3.99 (3.54, 4.39)	0.87 (−0.85, 2.30)	10.27 (2.71, 17.01)	1.10 (−1.79, 3.70)	4.62 (1.84, 7.14)
Total	3.88 (3.57, 4.18)	1.51 (0.61, 2.32)	4.42 (1.15, 7.50)	0.17 (−1.45, 1.68)	2.62 (0.71, 4.42)

BMI, body mass index (BMI > 40 kg/m^2); SES, socioeconomic status (<1st percentile of New York State); DM, diabetes; Systolic BP (systolic BP > 140 mm Hg).

and a higher SES compared to African Americans and Hispanics. African Americans and Hispanics had significantly higher rates of comorbidities including obesity, diabetes, and heart failure compared to non-Hispanic Whites. Also, African Americans had a higher mean systolic blood pressure and treatment for hypertension. There were 3663 patients whom had a clinical diagnosis of heart failure with a mean left ventricular ejection fraction (LVEF) of 48.5% (standard deviation of 16.6). Of these patients, 27.8% had heart failure with a reduced ejection fraction (LVEF < 35%).

Hazard ratios of risk factors for AF are presented in Table 2. Of the modifiable or risk factors examined, the presence of heart failure in African Americans and Hispanics was associated with a higher risk of incident AF compared to non-Hispanic Whites (HR = 2.12, 95% CI 1.88–2.39, p < 0.001; HR = 2.15, 95% CI 1.90–2.43, p < 0.001; HR = 1.48, 95% CI 1.28–1.71, p < 0.001; respectively). An elevated systolic blood pressure was also associated with a higher risk of incident AF in African Americans and Hispanics, but did not show any statistical association in non-Hispanic Whites (HR = 1.14, 95% CI 1.03–1.27, p = 0.009; HR = 1.18, 95% CI 1.06–1.31, p = 0.002, respectively). In addition, obesity in African Americans was associated with risk of incident AF, but not in other racial/ethnic groups.

Age and sex-adjusted population adjusted risk for examined risk factors are shown in Table 3 and Fig. 1. Again, the presence of heart failure and elevated systolic blood pressure in African Americans and Hispanics had a higher PAR for incident AF compared to non-Hispanic Whites, 7.93% and 7.66%, respectively. Obesity in African Americans and a lower SES in Hispanics also maintained a high PAR for incident AF compared to non-Hispanic Whites.

4. Conclusions

This is the largest study to date investigating the effect of modifiable risk factors by race/ethnicity for the association of incident AF in a racially and socioeconomically diverse inner-city population. We examined the association between race/ethnicity and modifiable risk factors for incident AF using a retrospective cohort. We confirm a higher incidence of atrial fibrillation in non-Hispanic Whites compared to African Americans and Hispanics. Moreover, we observed that African Americans and Hispanics had a higher population attributable risk of incident AF in the setting of heart failure or an elevated blood pressure compared to non-Hispanic Whites.

Our findings are consistent with previous studies including data reported by the cardiovascular health study demonstrating a 45% lower risk of AF in African Americans as compared to non-Hispanic Whites after adjusting for all risk factors of AF [10]. These findings were also confirmed by Alonso and colleagues, whom reported a 41% lower incidence of AF (95% CI: 0.08–0.62) in African Americans after adjusting for sex and age [17]. Few studies provide information on incidence AF in Hispanic populations, but review and statistical analysis of the Healthcare Cost and Utilization Project reported a lower AF risk in Hispanics (HR: 0.78; CI: 0.77–0.79. p < 0.001) as compared to non-Hispanic Whites [18].

In addition to these findings, we confirm that the distribution of risk factors for AF varied by race/ethnicity. For example, African Americans had a higher incidence of heart failure and treatment of hypertension

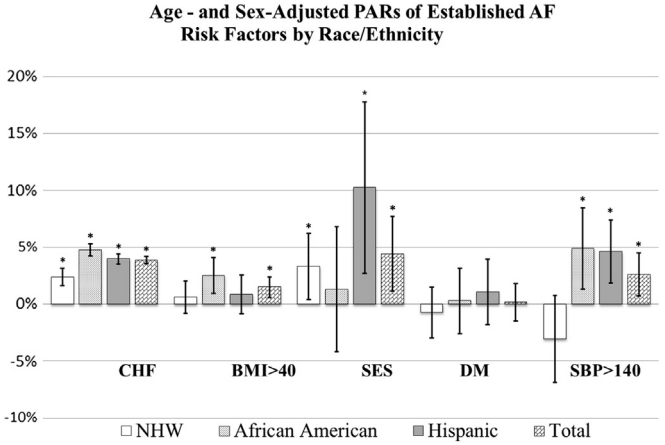

Fig. 1. Age- and sex-adjusted PARs of established AF risk factors by racial/ethnicity. Bar graph demonstrating population attributable risk for established modifiable risk factors for atrial fibrillation, stratified by race/ethnicity. HF, heart failure; BMI, body mass index > 40 kg/m^2; SES, socioeconomic status less than the 1st percentile; DM, diabetes mellitus, SBP, systolic blood pressure > 140 mm Hg; NHW, non-Hispanic White. Asterisk as indicated by (*) indicates statistical significance.

compared to non-African American cohorts, which was validated by prior reports [17,19]. Moreover, our study continues to demonstrate the "racial paradox" by showing that though non-Hispanic Whites have a higher incidence of AF, they have a lower burden of risk factors for AF.

4.1. Systolic blood pressure

We demonstrate potential differences in PAR estimates among racial and ethnic groups. One of the most significant differences was observed with elevation in systolic blood pressure, with 7.93% and 7.66% greater PAR estimates between African American and Hispanics, respectively, compared with non-Hispanics Whites. Among non-Hispanics Whites, the presence of an elevated systolic blood pressure was an independent predictor of incident AF as compared with African Americans or Hispanics. Though there is a small PAR difference between African Americans and Hispanics, which is likely not clinically relevant, there remains a larger discrepancy between these minority populations and non-Hispanic Whites, which may reflect underlying pathophysiological differences in substrate for incident AF.

Other large population studies have reported similar results. The MESA study included >50% non-Hispanic Whites subjects for PAR analysis of several AF risk factors. The greatest contributor among all groups was hypertension ranging from 22% in non-Hispanic whites to 44% and 46% in Hispanics and Chinese, respectively. The authors concluded that a larger proportion of AF events appear to be attributable to hypertension among a minority population compared with non-Hispanic Whites [20]. This finding was also confirmed in the REGARDS [21] and the Women's Health Initiative study [10].

Significantly lower rates of hypertension control might explain elevated PAR estimates in non-Whites compared to Whites. The NHANES [22] study noted significantly lower rates of hypertension control among African Americans. Moreover, the SPRINT trial [23] showed that despite similarities in systolic blood pressure at baseline by race, African Americans were more likely to have elevated blood pressures than non-African Americans, despite being prescribed slightly more anti-hypertensive medications. Of note, these studies were limited as they lacked a large Hispanic population. Regardless, given similar findings, improving awareness, control and management of hypertension in non-Whites may result in improved treatment, and control rates of AF.

4.2. Heart failure

Another observable difference in PAR estimates includes the presence of heart failure. African Americans and Hispanics had higher PAR estimates compared to their non-Hispanic counterparts. The hazard ratios for the presence of heart failure for risk of incident AF were significantly higher in African Americans and Hispanics compared to non-Hispanic Whites.

However, it remains unclear why the presence of heart failure has a higher hazard ratio and PAR with AF in minorities. One explanation may be a racial/ethnic-related difference in the prevalence of hypertension and diabetes mellitus. These differences in comorbidities are likely related to socioeconomic and behavioral characteristics, and may include disparities in access to and quality of health care, as shown in prior heart failure studies [24,25] as well as our analyses. Also, possible racial/ethnic differences in susceptibility to heart failure owing to underlying genetic and biological factors cannot be completely excluded [26]. Intrinsic racial differences in atrial membrane stability, atrial conduction pathways, or genetic polymorphisms leading to different susceptibility to the development of AF may play a role. For example, polymorphisms have been found to be associated with racial differences in risk for heart failure [27].

4.3. Obesity

A higher BMI was significantly associated with incident AF, independent of other modifiable risk factors, with a 27% increased risk if the BMI was >40 kg/m^2, which was maintained only in the African American cohort. Other studies have found similar results with an elevated hazard ratio for BMI on incident AF [28] and higher PAF estimates [10] in African Americans. Obesity has been previously described as an important risk factor for AF [29–31], and the effects are thought to be mediated through increased left atrial size [29] or presence of pericardial fat [32].

An obesity-hypertension relationship may explain the elevated PAR estimates in African Americans. Hypertension is strongly associated with obesity. Moreover, the prevalence of obesity among patients with hypertension is much higher than the population prevalence of obesity [33]. Given these relationships and our studies' findings, there appears to be an added effect of race on the obesity-hypertension relationship. Thus, it is likely that elevated PAR estimates for obesity are exclusively demonstrated in African Americans given their high prevalence of both hypertension and obesity as comorbidities.

Our study suggest that BMI, and by virtue obesity, appears to play a different role in various racial/ethnic groups. Given the obesity epidemic, these findings are yet another reason more attention should be paid to population-based efforts to control obesity and to further study the mechanisms linking obesity and AF.

4.4. Socioeconomic status

Additional differences included a higher PAR in Hispanics compared to African Americans and non-Hispanic Whites, at 8.97% and 6.96% respectively. In our study, Hispanics maintained a significantly lower SES score compared to other racial and ethnic groups. Moreover, there exists no statistical association between socioeconomic status and incident AF in the African American cohort. Given a lower socioeconomic status in Hispanics compared to all other racial/ethnic groups, our results may suggest that differences in access to, use of health care, or prior diagnosis of AF (ascertainment bias) may account in part for our findings.

Several studies have identified risk factors, which can be modified for AF outcomes. The Atherosclerosis in Communities (ARIC) performed by Huxley et al. reported that approximately 56% of AF cases could be attributed to one or more sub-optimally controlled or elevated risk factors. Hypertension was the most contributory risk factor with its PAR being 22%. Similarly, other risk factors like elevated body mass index, diabetes mellitus and smoking had PARs of 13%, 3.1% and 9.8% respectively [34]. These risk factors not only contribute to the increased incidence of AF independently, but also have been reported to increase recurrence of AF after catheter ablation [29,35–37]. In the study conducted by Pathak and colleagues, the group of AF patients who chose to participate in risk factor modification after their ablation procedure had greater decline in weight, better control of diabetes and hypertension. They had a longer arrhythmia free period after an ablation procedure (32.9%) as compared to control group (9.7%) $p < 0.001$ [38]. Several studies propose that cardiac risk factors are closely associated with the remodeling of the atria leading to formation of underlying substrate causing AF development, progression and likely recurrence in the event of sub-optimal control of risk factors [38–40]. These findings further strengthen the argument that modification of risk factors including antihypertensive therapy, weight loss and management of heart failure can improve AF outcomes.

There are significant public health implications of our findings. It is important to note that Hispanics, along with African Americans, tend to share a similar disease burden profile of AF compared to non-Hispanic Whites [8]. Racial and ethnic variability of PAR shows that hypertension and heart failure have a higher correlative risk to incident AF among the non-White population. As such, we suggest that healthcare

strategies should focus on blood pressure control and risk factor modification as ways to decrease incident AF in heart failure in these select populations. This study makes an important contribution to existing literate as it presents new information showing the important contribution of heart failure, and confirms the contribution of hypertension, to risk of incident AF in the non-White population.

4.5. Limitations

We acknowledge limitations to our study. First, it is possible that patients were enrolled in our study with a previous diagnosis of AF, but did not demonstrate an irregular rhythm on ECG and were misclassified. In addition, segments of our population were also excluded due to incomplete patient data (mostly consisting of height/weight), which could have biased our results. We also did not include patients with sleep apnea or pulmonary disease (i.e. chronic obstructive pulmonary disease (COPD)) in this study.

5. Conclusion

The findings of the current study suggest that modifiable risk factors play an important role in risk of incident AF, and vary by race/ethnicity. A higher PAR estimate in African American and Hispanics were observed for elevated systolic blood pressure and heart failure. To our knowledge, this is the largest study to date investigating the effect of modifiable risk factors by race/ethnicity for the risk of incident AF in a racially and socioeconomically diverse, inner-city population. The authors hope that identification of these modifiable risk factors, particularly hypertension and heart failure in minorities may assist in targeting better prevention therapies of atrial fibrillation from a public health perspective.

Conflicts

None of the authors have any conflict of interest. None of the authors received any compensation for their work on this manuscript.

Acknowledgements

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

[1] A. Odutayo, C.X. Wong, A.J. Hsiao, S. Hopewell, D.G. Altman, C.A. Emdin, Atrial fibrillation and risks of cardiovascular disease, renal disease, and death: systematic review and meta-analysis, BMJ 354 (2016), i4482.
[2] M.H. Kim, S.S. Johnston, B.C. Chu, M.R. Dalal, K.L. Schulman, Estimation of total incremental health care costs in patients with atrial fibrillation in the United States, Circ. Cardiovasc. Qual. Outcomes 4 (2011) 313–320.
[3] A.S. Go, E.M. Hylek, K.A. Phillips, Y. Chang, L.E. Henault, J.V. Selby, D.E. Singer, Prevalence of diagnosed atrial fibrillation in adults, JAMA 285 (2001) 6.
[4] K.L. Hong, B.M. Glover, The impact of lifestyle intervention on atrial fibrillation, Curr. Opin. Cardiol. 33 (2018) 14–19.
[5] R.B. Schnabel, Can we predict the occurrence of atrial fibrillation? Clin. Cardiol. 35 (2012) 5.
[6] R.B. Schnabel, T. Aspelund, G. Li, L.M. Sullivan, A. Suchy-Dicey, T.B. Harris, M.J. Pencina, R.B. D'Agostino, D. Levy, W.B. Kannel, T.J. Wang, R.A. Krommal, P.A. Wolf, G.L. Burke, L.J. Launer, R.S. Vasan, B.M. Psaty, E.J. Benjamin, V. Gudnason, S.R. Heckbert, Validation of an atrial fibrillation risk algorithm in whites and African Americans, Arch. Intern. Med. 170 (2010) 9.
[7] L. Wang, J. Southerland, K. Wang, B.A. Bailey, A. Alamian, M.A. Stevens, Y. Wang, Ethnic differences in risk factors for obesity among adults in California, the United States, J. Obes. 2017 (2017) 2427483.
[8] E. Shulman, F. Kargoli, P. Aagaard, E. Hoch, L. Di Biase, J. Fisher, J. Gross, S. Kim, K.J. Ferrick, A. Krumerman, Socioeconomic status and the development of atrial fibrillation in Hispanics, African Americans and non-Hispanic whites, Clin. Cardiol. 40 (9) (2017) 770–776.
[9] E. Shulman, F. Kargoli, P. Aagaard, E. Hoch, L. Di Biase, J. Fisher, J. Gross, S. Kim, A. Krumerman, K.J. Ferrick, Validation of the Framingham heart study and CHARGE-AF risk scores for atrial fibrillation in Hispanics, African-Americans, and non-Hispanic whites, Am. J. Cardiol. 117 (2016) 76–83.
[10] P.N. Jensen, E.L. Thacker, S. Dublin, B.M. Psaty, S.R. Heckbert, Racial differences in the incidence of and risk factors for atrial fibrillation in older adults: the cardiovascular health study, J. Am. Geriatr. Soc. 61 (2013) 276–280.
[11] Facts Q, US Census Bureau, 2013 (Retrieved Jan 2013).
[12] IEC IS, Particular Requirements for Safety, Including Essential Performance, of Recording and Analyzing Single Channel and Multichannel Electrocardiographs, 2003.
[13] F. Bogun, D. Anh, G. Kalahasty, E. Wissner, C. Bou Serhal, R. Bazzi, W.D. Weaver, C. Schuger, Misdiagnosis of atrial fibrillation and its clinical consequences, Am. J. Med. 117 (2004) 636–642.
[14] M.E. Guglin, D. Thatai, Common errors in computer electrocardiogram interpretation, Int. J. Cardiol. 106 (2006) 232–237.
[15] K. Poon, P.M. Okin, P. Kligfield, Diagnostic performance of a computer-based ECG rhythm algorithm, J. Electrocardiol. 38 (2005) 235–238.
[16] A. Hajat, A.V. Diez-Roux, S.D. Adar, A.H. Auchincloss, G.S. Lovasi, M.S. O'Neill, L. Sheppard, J.D. Kaufman, Air pollution and individual and neighborhood socioeconomic status: evidence from the Multi-Ethnic Study of Atherosclerosis (MESA), Environ. Health Perspect. 121 (2013) 1325–1333.
[17] A. Alonso, S.K. Agarwal, E.Z. Soliman, M. Ambrose, A.M. Chamberlain, R.J. Prineas, A.R. Folsom, Incidence of atrial fibrillation in whites and African-Americans: the Atherosclerosis Risk in Communities (ARIC) study, Am. Heart J. 158 (2009) 111–117.
[18] T.A. Dewland, J.E. Olgin, E. Vittinghoff, G.M. Marcus, Incident atrial fibrillation among Asians, Hispanics, blacks, and whites, Circulation 128 (2013) 2470–2477.
[19] J.A. Cutler, P.D. Sorlie, M. Wolz, T. Thom, L.E. Fields, E.J. Roccella, Trends in hypertension prevalence, awareness, treatment, and control rates in United States adults between 1988–1994 and 1999–2004, Hypertension 52 (2008) 818–827.
[20] C.J. Rodriguez, E.Z. Soliman, A. Alonso, K. Swett, P.M. Okin, D.C. Goff Jr., S.R. Heckbert, Atrial fibrillation incidence and risk factors in relation to race-ethnicity and the population attributable fraction of atrial fibrillation risk factors: the multi-ethnic study of atherosclerosis, Ann. Epidemiol. 25 (2015) 71–76 (76 e71).
[21] W.T. O'Neal, S.E. Judd, N.A. Limdi, W.F. McIntyre, D.O. Kleindorfer, M. Cushman, V.J. Howard, G. Howard, E.Z. Soliman, Differential impact of risk factors in blacks and whites in the development of atrial fibrillation: the Reasons for Geographic And Racial Differences in Stroke (REGARDS) study, J. Racial Ethn. Health Disparities 4 (4) (2017) 718–724.
[22] I. Hajjar, T.A. Kotchen, Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988–2000, JAMA 290 (2003) 199–206.
[23] C.H. Still, T.E. Craven, B.I. Freedman, P.N. Van Buren, K.M. Sink, A.A. Killeen, J.T. Bates, A. Bee, G. Contreras, S. Oparil, C.M. Pedley, B.M. Wall, S. White, D.M. Woods, C.J. Rodriguez, J.T. Wright Jr., Group SSR, Baseline characteristics of African Americans in the systolic blood pressure intervention trial, J. Am. Soc. Hypertens. 9 (2015) 670–679.
[24] M. Alexander, K. Grumbach, J. Selby, A.F. Brown, E. Washington, Hospitalization for congestive heart failure. Explaining racial differences, JAMA 274 (1995) 1037–1042.
[25] H. Bahrami, R. Kronmal, D.A. Bluemke, J. Olson, S. Shea, K. Liu, G.L. Burke, J.A. Lima, Differences in the incidence of congestive heart failure by ethnicity: the multi-ethnic study of atherosclerosis, Arch. Intern. Med. 168 (2008) 2138–2145.
[26] E. van Rooij, L.B. Sutherland, X. Qi, J.A. Richardson, J. Hill, E.N. Olson, Control of stress-dependent cardiac growth and gene expression by a microRNA, Science 316 (2007) 575–579.
[27] K.M. Small, L.E. Wagoner, A.M. Levin, S.L. Kardia, S.B. Liggett, Synergistic polymorphisms of beta1- and alpha2C-adrenergic receptors and the risk of congestive heart failure, N. Engl. J. Med. 347 (2002) 1135–1142.
[28] R.B. Schnabel, T. Aspelund, G. Li, Validation of an atrial fibrillation risk algorithm in whites and African Americans, Arch. Intern. Med. 170 (2010) 9.
[29] T.J. Wang, H. Parise, D. Levy, R.B. D'Agostino Sr., P.A. Wolf, R.S. Vasan, E.J. Benjamin, Obesity and the risk of new-onset atrial fibrillation, JAMA 292 (2004) 2471–2477.
[30] A.S. Gami, D.O. Hodge, R.M. Herges, E.J. Olson, J. Nykodym, T. Kara, V.K. Somers, Obstructive sleep apnea, obesity, and the risk of incident atrial fibrillation, J. Am. Coll. Cardiol. 49 (2007) 565–571.
[31] E. Shulman, J.J. Chudow, T. Shah, K. Shah, A. Peleg, D. Nevelev, F. Kargoli, L. Zaremski, C. Berardi, A. Natale, J. Romero, L. Di Biase, J. Fisher, A. Krumerman, K.J. Ferrick, Relation of body mass index to development of atrial fibrillation in Hispanics, blacks, and non-Hispanic whites, Am. J. Cardiol. 121 (10) (2018) 1177–1181.
[32] G. Thanassoulis, J.M. Massaro, C.J. O'Donnell, U. Hoffmann, D. Levy, P.T. Ellinor, T.J. Wang, R.B. Schnabel, R.S. Vasan, C.S. Fox, E.J. Benjamin, Pericardial fat is associated with prevalent atrial fibrillation: the Framingham heart study, Circ. Arrhythm. Electrophysiol. 3 (2010) 345–350.
[33] N. Cossrow, B. Falkner, Race/ethnic issues in obesity and obesity-related comorbidities, J. Clin. Endocrinol. Metab. 89 (2004) 2590–2594.
[34] R.R. Huxley, F.L. Lopez, A.R. Folsom, S.K. Agarwal, L.R. Loehr, E.Z. Soliman, R. Maclehose, S. Konety, A. Alonso, Absolute and attributable risks of atrial fibrillation in relation to optimal and borderline risk factors: the Atherosclerosis Risk in Communities (ARIC) study, Circulation 123 (2011) 1501–1508.
[35] K. Jongnarangsin, A. Chugh, E. Good, S. Mukerji, S. Dey, T. Crawford, J.F. Sarrazin, M. Kuhne, N. Chalfoun, D. Wells, W. Boonyapisit, F. Pelosi Jr., F. Bogun, F. Morady, H. Oral, Body mass index, obstructive sleep apnea, and outcomes of catheter ablation of atrial fibrillation, J. Cardiovasc. Electrophysiol. 19 (2008) 668–672.
[36] D. Conen, U.B. Tedrow, B.A. Koplan, R.J. Glynn, J.E. Buring, C.M. Albert, Influence of systolic and diastolic blood pressure on the risk of incident atrial fibrillation in women, Circulation 119 (2009) 2146–2152.
[37] U.B. Tedrow, D. Conen, P.M. Ridker, N.R. Cook, B.A. Koplan, J.E. Manson, J.E. Buring, C.M. Albert, The long- and short-term impact of elevated body mass index on the risk of new atrial fibrillation the WHS (women's health study), J. Am. Coll. Cardiol. 55 (2010) 2319–2327.

[38] R.K. Pathak, M.E. Middeldorp, D.H. Lau, A.B. Mehta, R. Mahajan, D. Twomey, M. Alasady, L. Hanley, N.A. Antic, R.D. McEvoy, J.M. Kalman, W.P. Abhayaratna, P. Sanders, Aggressive risk factor reduction study for atrial fibrillation and implications for the outcome of ablation: the ARREST-AF cohort study, J. Am. Coll. Cardiol. 64 (2014) 2222–2231.

[39] D.H. Lau, L. Mackenzie, D.J. Kelly, P.J. Psaltis, A.G. Brooks, M. Worthington, A. Rajendram, D.R. Kelly, Y. Zhang, P. Kuklik, A.J. Nelson, C.X. Wong, S.G. Worthley, M. Rao, R.J. Faull, J. Edwards, D.A. Saint, P. Sanders, Hypertension and atrial fibrillation: evidence of progressive atrial remodeling with electrostructural correlate in a conscious chronically instrumented ovine model, Heart Rhythm. 7 (2010) 1282–1290.

[40] H.S. Abed, C.S. Samuel, D.H. Lau, D.J. Kelly, S.G. Royce, M. Alasady, R. Mahajan, P. Kuklik, Y. Zhang, A.G. Brooks, A.J. Nelson, S.G. Worthley, W.P. Abhayaratna, J.M. Kalman, G.A. Wittert, P. Sanders, Obesity results in progressive atrial structural and electrical remodeling: implications for atrial fibrillation, Heart Rhythm. 10 (2013) 90–100.